The Supernormal Phase of Intraventricular Conduction

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The supernormal phase of conduction is a short period of paradoxically improved conduction which may occur during an early phase of the recovery cycle of cardiac conducting tissue. The situation is paradoxical, for the improved conduction occurs during a short critical early period, while later impulses are blocked or have greater conduction delays. The term is, in a sense, a misnomer since conduction is not supernormal but merely a temporary improvement of a depressed state of conduction. Indeed, the supernormal phase of conduction does not occur in the normal heart but only when conductivity is depressed. The phase of supernormality is usually very short and only lasts a few hundredths of a second. Its position in the cardiac cycle is usually coincident with the U wave or the distal limb of the T wave.

The supernormal phase of conduction has been demonstrated in the AV node, but could theoretically occur at any site with an actual or potential conduction delay. The following cases demonstrate the supernormal phase of intraventricular conduction.

CASE REPORTS

Case 1. The electrocardiogram was recorded from a 69-year-old woman with a toxic adenoma of the thyroid gland. She also had mild aortic incompetence and aortic stenosis and was on maintenance digitalis.

The tracings (Fig. 1) are simultaneous recordings of leads V1 and V2. Strips A and B are continuous. Strips C to E are also continuous and were taken from a later section of the same recording. These tracings show atrial fibrillation with a variable ventricular response. The R–R intervals range from 45 to 170†. QRS complexes that terminate relatively short cycles—cycles with R–R intervals that range from 45 to 106—have a left bundle-branch block pattern. QRS complexes that terminate long cycles—cycles with R–R intervals that range from 106 to 170—have a normal QRS pattern.

The fluctuation between normal and abnormal intraventricular conduction indicates that the left bundle-branch block is a manifestation of phasic aberrant ventricular conduction (Schamroth and Chesler, 1963)—the transient, intermittent, or isolated intraventricular conduction of a supraventricular impulse. This phenomenon is basically due to unequal refractory periods of the bundle-branches. When this occurs, a relatively early impulse may encounter a responsive right bundle-branch and a refractory left bundle-branch, and will consequently be conducted with a left bundle-branch block pattern.

Longer pauses permit full recovery of both bundle-branches, and late impulses are therefore conducted normally. Prematurity is thus a major factor in the genesis of aberrant ventricular conduction and prematurity must be considered pari passu with the duration of refactoriness. Refractoriness varies directly with the duration of the preceding cycle—the longer the preceding cycle (the preceding R–R interval), the longer the subsequent refractory period, and vice versa (Trendelenburg, 1903; Mines, 1913; Lewis, Drury, and Bulger, 1921). This principle also applies when the refractory periods of the bundle-branches are unequal, i.e. the refractory period of each bundle-branch will also be shortened or lengthened by preceding short or long intervals; and this may effect the conduction of a subsequent early impulse. For example, a premature impulse when preceded by a long cycle may encounter a refractory bundle-branch and be conducted with aberration, whereas an impulse of the same prematurity, when preceded by a short cycle, may find both bundle-branches responsive and will consequently be conducted normally. These principles are obviously of major importance in rhythms such as atrial fibrillation where cycle lengths vary considerably. Any comparison of the conduction sequences of different impulses under such circumstances must therefore take into account the effect of prematurity as well as the effect of the preceding cycle lengths. This comparison is facilitated by plotting each R–R interval against its preceding R–R interval (Fig. 2). The parameters of both prematurity and preceding cycle lengths of different impulses can now be compared. For example, consider a specific point on the graph, reflecting an impulse of certain prematurity that is preceded by a certain cycle length (illustrated as

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† All time intervals are expressed in hundredths of a second, i.e. 45 = 45 hundredths of a second.
FIG. 1.—Electrocardiogram (Case 1). Strips A and B are continuous and simultaneous recordings of leads V1 and V2. Strips C to E are continuous and simultaneous recordings of leads V1 and V2 taken from a later section of the same recording. Long cycles are terminated by normal QRS complexes (black dots). Short cycles are terminated by complexes with a left bundle-branch block pattern. Two short cycles are paradoxically terminated by normal QRS complexes (labelled X). See text.

The single black point on the graph in Fig. 2). Any later impulse which is preceded by a shorter cycle length—down and to the right of the graph—will encounter tissue that is less refractory and should therefore be conducted more normally. Contrariwise, impulses that occur more prematurely and are preceded by longer R-R intervals—upwards and to the left in the graph (in the shaded area)—will encounter tissue that is more refractory, and conduction should consequently be more abnormal. Comparison of other regions of the graph with the specific point is not valid. Thus, impulses that occur earlier but are preceded by shorter R-R intervals—down and to the left—may still be conducted normally since the shorter preceding R-R interval will shorten the subsequent refractory period and thus counteract the effect of prematurity. Impulses that occur later but are preceded by longer cycles—up and to the right in the graph—may still be conducted abnormally since the long preceding cycle will lengthen the subsequent refractory period and thus tend to favour aberrant ventricular conduction. In other words, the parameters of prematurity and preceding cycle length counteract each other in positions up and to the right, and down and to the left of the specific point. Comparison of impulses in these regions with the specific point is therefore difficult and probably invalid. However, the parameters of prematurity and preceding cycle length complement each other in positions up and to the left and down and to the right of the specific point. Comparison of impulses in these regions with the specific point is therefore valid.

Further analysis of the electrocardiograms of Case 1 reveals two exceptions to these aforementioned principles. There are two short cycles—with R-R intervals
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of 53 and 55—which are terminated by QRS complexes that unexpectedly have a normal QRS configuration (complexes labelled X in strips C and D). Furthermore, when each R-R interval of this tracing is plotted against its preceding R-R interval (Fig. 3), it is clear that many of the later impulses which are preceded by shorter R-R intervals and which should therefore be conducted more normally, are, in fact, conducted aberrantly. For example, one of the premature impulses referred to above terminates an R-R interval of 53 and is preceded by an R-R interval of 83. Many impulses which occur later and are preceded by shorter impulses (open circles within the area bounded by the dotted lines) and which should be conducted normally, are, in fact, conducted aberrantly.

Thus, conduction through the left bundle-branch is paradoxically better at a critical early stage of recovery. It will be observed that normal conduction through the left bundle-branch occurs during 2 periods: (1) when impulses are very late, and (2) when impulses are very early—during a short critical period. Impulses that occur between these 2 extremes are conducted abnormally.

Case 2. The electrocardiogram (Fig. 4—a continuous strip of lead V1) was recorded from a 37-year-old man with mitral valvular disease who was on maintenance digitalis. The tracing shows atrial fibrillation with a variable ventricular response. The R-R intervals range from 36 to 156. QRS complexes that terminate long cycles, i.e. cycles that range from 108 to 156, have a normal configuration. QRS complexes that terminate short cycles, i.e. cycles that range from 36 to 98, have a
FIG. 4.—Electrocardiogram (Case 1)—a continuous strip of lead V1—showing atrial fibrillation with a variable ventricular response. Long cycles are terminated by normal QRS complexes. Short cycles are terminated by QRS complexes with a right bundle-branch block pattern. Three short cycles are paradoxically terminated by normal QRS complexes labelled a, b, and c. See text.

right bundle-branch block pattern. This is also reflected in the graph (Fig. 5) where each R–R interval is plotted against its preceding R–R interval. The transition from normal to abnormal intraventricular conduction is marked by a period ranging from 98 to 108 during which conduction may be either normal or abnormal. There are 3 exceptions to the aforementioned distribution. Three QRS complexes are recorded prematurely (terminating cycles of 81, 85 and 90) and paradoxically have a normal configuration. These complexes are labelled a, b, and c in the electrocardiogram (Fig. 4) and are reflected as points a, b, and c in the graph (Fig. 5). Reference to any one of these specific points on the graph reveals that there are later impulses which are preceded by shorter R–R intervals which have abnormal intraventricular conduction, e.g. the open circles within the squares bounded by the dotted lines. These impulses should be conducted more normally but are in fact conducted aberrantly. A transient improvement of conduction with the blocked right bundle-branch thus occurs paradoxically, during a short critical period at an early stage of recovery.

DISCUSSION

The supernormal phase of AV conduction has been well documented (Lepeschkin and Kimura, 1963; Pick, Langendorf, and Katz, 1962), approximately 50 cases having been published to date. The condition is probably more common than this relatively small published number would indicate. The supernormal phase of intraventricular conduction, however, is exceedingly rare. Very few cases have been reported, and most of these are subject to alternative explanation. Contro, Magri, and Natali (1956) reported two cases where a dominant rhythm with a bundle-branch block pattern was
interrupted by premature beats with a normal QRS configuration. This manifestation could alternatively have been due to septal origin of the premature beats below the region of block, or conduction of the premature impulses through accessory pathways. Septal origin of premature beats below the region of block was postulated in three similar cases described by Simon and Langendorf (1944). They suggested the alternative but less likely diagnosis of the supernormal phase of intraventricular conduction for one of these cases. Pick and Fishman (1950) reported a case of high-grade AV block with a subsidiary AV nodal pacemaker whose impulses were conducted with typical right bundle-branch block pattern. The rhythm was complicated by premature (capture) beats which had a normal QRS configuration. This was interpreted as the supernormal phase of intraventricular conduction.

Scherf and Scharf (1948) reported a case where the first beat of a Wenckebach sequence was conducted with a bundle-branch block pattern, whereas the remaining beats of the sequence had a normal QRS configuration. The normal conduction was thought to result from conduction during the supernormal phase of the depressed bundle-branch. Scherf and Scharf postulated a similar mechanism in a case described by von Hoesslin (1923). Burchnell (1949) reported a case of interference-dissociation where the capture beats had either a right or left bundle-branch block pattern. This was dependent upon the R–P interval—the time relation between the R wave of the idionodal pacemaker and the P wave of the atrial pacemaker, and was explained on the basis of different recovery rates of the two bundle-branches. An alternative explanation of an intraventricular supernormal phase of recovery was considered less likely.

The uncertainty of the postulated supernormal mechanism in the aforementioned cases is due in part to the fact that these cases reflect an apparent early improvement of intraventricular conductivity whereas all later impulses are blocked, i.e. show the bundle-branch block pattern. The two cases described here, however, show normal intraventricular conduction during both early and late phases of the cardiac cycle, whereas an intermediate period

![Fig. 5.—Case 2. Graphic representation of each R–R interval against its preceding R–R interval. See text.](http://heart.bmj.com/br-heart-j-first-published-as-10.1136/hrt.31.3.337-on-1-may-1969/downloaded-from)
reflects the impaired intraventricular conduction. This establishes a truly paradoxical situation and invalidates many of the cogent arguments brought against the diagnosis of the supernormal phase of intraventricular conduction—a mechanism that would appear to be the most likely explanation in these two cases. A phenomenon that moreover may occur in conduction through either the left bundle-branch (Case 1) or the right bundle-branch (Case 2).

SUMMARY

Two cases of atrial fibrillation with phasic aberrant ventricular conduction are described. Early supraventricular impulses were conducted normally. Occasional exceptions to this principle occurred, and consisted of unexpected normal intraventricular conduction of early impulses. This phenomenon is explained on the basis of the supernormal phase of intraventricular conduction. The significance of the preceding cycle length in relation to aberration is discussed, and a method is described whereby both the parameters of prematurity and preceding cycle length of different impulses can be compared.

REFERENCES


